with death or rMI). We then validated this model in a pre-specified subgroup of patients from the GRACE registry who met criteria for inclusion in the TIMI 11B trial (n=11505).

Results: 8 independent predictors of in-hospital death or rMI found upon presentation were identified (table). The C-statistic for the model was 0.70. Using the model in the TIMI-like subgroup, the C-statistic was 0.70 as well. We also created a simplified, additive scoring system for weighting each predictive variable which can be used to estimate the risk of death or rMI by using a pocket prediction card or handheld PDA.

	OR (95% CI)
Age (per 10 yr increase)	1.2(1.13 – 1.22)
Heart Rate (per 30 bpm increase)	1.1(1.03 – 1.18)
Systolic Blood Pressure (per 20 mmHg decrease)	1.1(1.04 – 1.15)
Initial serum creatinine (per 1 mg/dl increase)	1.1(1.04 – 1.15)
Initial cardiac enzyme elevation	1.8(1.64 – 2.00)
Killip class (per increase in 1 class)	1.4(1.34 – 1.56)
Cardiac arrest at admission	3.0(2.21 – 3.93)
ST segment deviation	2.1(1.89 – 2.34)

Conclusions:
This GRACE model shows a strong ability to predict the occurrence of the combined endpoint of death or rMI in all patients presenting with ACS and lends itself to incorporation in a handheld PDA or pocket prediction tool for bedside use.

POSTER SESSION

1096 Basic Myocardial Infarction

Monday, March 31, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: Noon-1:00 p.m.

1096-103 Molecular Basis of Electrical Remodeling in the Infarct Zone

Sumeet S. Chugh, Stacey Whitesel, Srinivasa Nagalla, Oregon Health & Science University, Portland, OR

Background: Altered electrophysiologic properties of infarcted vs non-infarcted myocardium cause reentrant ventricular arrhythmias. A global assessment of comparative differential gene expression would be useful, but has not been performed.
Methods: We therefore, used cDNA microarray gene profiling to characterize differential cardiac gene expression patterns in infarcted vs non-infarcted myocardium of rats (n=9) with surgically induced acute myocardial infarction. LV was harvested and infarcted vs non-infarcted myocardium separated 1, 4 and 5 weeks post-surgery. Total RNA were extracted and pooled for 3 samples at each time point. Fluorescent, labeled complex cDNA probes were generated by reverse transcription and hybridized to microarrays containing 13,824 sequence-verified, non-redundant rodent cDNA clones.
Results: Significant LV infarction was observed in all animals (≥30% LV), with progression to severe dilated cardiomyopathy by 4 wks. Of total genes profiled, n=4, n=42 and n=694 exhibited significant differential expression (Arraystat) in infarcted vs non-infarcted myocardium at 1, 4 and 5 weeks respectively. The earliest differential expression was identified in the T-type voltage-dependent Ca^{2+} channel α 1H subunit (Infarcted 5-fold increase) and mitochondrial Cl^- intracellular channel 4 (Infarcted 5-fold increase). Cell communication, cell growth & maintenance as well as cell death genes exhibited increasing differential expression over time.
Conclusion: Extent of differential gene expression correlated with time period following MI. Early and significant alterations were identified in T-type Ca^{2+} channel and mitochondrial intracellular Cl^- channel.

1096-104 Surface Area of Perfusion Defects as the Determinant of the Effect of Coronary Microembolization: A Micro-Computed Tomography Study

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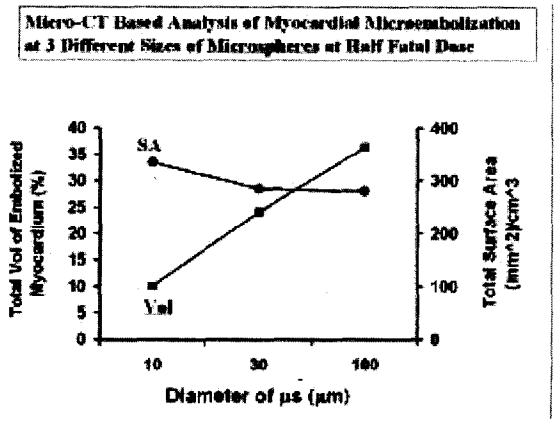
Background: In Myocardial infarction due to occlusion of an epicardial artery, the impairment of the left ventricular function and clinical outcome is proportional to the volume of infarcted myocardium. As this relationship does not apply in myocardial microembolization (see Figure), we hypothesize that the total surface area of the myocardial microinfarcts is the more important determinant of the clinical outcome.
Materials and Methods: We induced myocardial perfusion defects (MPD) by injecting polymer microspheres (μsph, 10, 30 or 100 μm diameter) at 3 doses (1/8, 1/4 or 1/2 fatal dose) into the LAD or LCX of 8 anesthesized pigs (30 ±1 kg). 3D micro-CT images, with 20μm on-a-side cubic voxels, were generated of postmortem transmural biopsies (~ 1 cm3) from the embolized myocardium. From these images we calculated the individual and total surface area, and volume, of the embolized territories, for each size and each dose of μsph.
Results: The total volume of the non-perfused territories was logarithmically related to the μsph diameter and the total number of MPD was 203, 145 and 76 for the 10, 30, and 100 μm μsph, respectively. However, the total surface area of the same non-perfused territories was essentially constant, independent of the μsph diameter, as illustrated in the Figure.
Conclusion: The total surface area of the embolized territories is directly related to the fatal impact of myocardial microembolization.



1096-105 Mechanical Left Ventricular Unloading Immediately Prior to Reperfusion Reduces Infarct Size

Hela C. Achour, Fernando Boccolandro, Patty Felli, James Amirian, Ricchard W. Smalling, University of Texas Houston Medical School, Houston, TX

Aim- To test the hypothesis that unloading the left ventricle (LV) just prior to reperfusion limits reperfusion related myocardial injury and provides infarct size reduction compared with LV unloading post reperfusion and reperfusion alone. Methods- Twenty-four mongrel dogs were subjected to 2 hours of left anterior descending artery occlusion and 4 hours of reperfusion. A trans-valvular left ventricular assist device (TV LVAD) (Medtronic, Inc) was inserted just prior to reperfusion and maintained during the rest of the experiment in group-1. In group-2 the TV LVAD was inserted and activated just after reperfusion. Group-3 (controls) was subjected to reperfusion alone with no LV support. Pressure catheters were placed in the left atrial appendage, LV apex, and ascending aorta for hemodynamic measurements. Regional myocardial blood flow (RMBF), infarct size, post-reperfusion end diastolic wall thickness (EDWT) in the ischemic region and electron microscopy in the central ischemic zone and control regions were determined. Measurements were made at baseline, 2 hours after coronary occlusion, with reperfusion and at 2, 3 and 4 hours after reperfusion. Results- The hemodynamic data at baseline were similar in the 3 groups. Myocardial infarct size expressed as percentage of area at risk was significantly reduced in group-1 compared to the control group (34.69 ± 4.62% vs 54.58 ± 9.23% respectively, P=0.011) and to group-2 (34.69 ± 4.62% vs 51.51 ± 14.04% respectively, p<0.05). At 4 hours of reperfusion, absolute RMBF in the ischemic zone was slightly higher in group 1 compared to controls, and significantly higher than in group-2 (p=0.04). EDWT tended to return to baseline level in group-1 compared to both controls and group-2, which demonstrated a significant post-reperfusion increase in EDWT and contraction band necrosis in the central ischemic region of these groups. There was a good correlation between the increase in post-reperfusion myocardial wall thickness in the ischemic region and the extent of myocardial infarction. Conclusions- LV unloading prior to, but not after reperfusion, reduces the extent of myocardial injury in canine hearts subjected to 2h of LAD occlusion and 4 hours of reperfusion.

1096-106 Nonangiographically Evident Collateral Flow During Primary Percutaneous Transluminal Coronary Angioplasty Favors Contractile Recovery

Anna Sonia Petronio, Ugo Limbruno, Giovanni Amoroso, Roberta Rossini, Marco De Carlo, Andrea Micheli, Luca Pataleo, Caterina Palagi, Paolo Caravelli, Mario Mariani, University of Pisa, Pisa, Italy

BACKGROUND: Recent observations suggest that in patients (pts) undergoing primary PTCA for acute myocardial infarction (AMI), time to treatment has a limited impact on prognosis when greater than 2 hours. Other factors may influence myocardial salvage. The aim of the study was to evaluate if the presence of a preserved and adequate collateral network may limit microvascular damage, and consequently prevent left ventricular dysfunction, during the occlusion of an epicardial coronary artery in pts undergoing primary PTCA with a time to reperfusion > 2 hours.
METHODS: We studied 12 consecutive pts with first uncomplicated AMI, successfully treated with primary PTCA with stenting. All pts had Thrombolysis In Myocardial Infarction (TIMI) grade 0-1 in the infarct-related artery (IRA). Standard abciximab treatment was used in all procedures. Intracoronary pressure measurements were performed immediately before stent implantation in all pts, using a PressureWire System. Collateral flow was assessed as fractional collateral flow (FCF) , determined as the ratio of coronary wedge pressure to mean aortic pressure simultaneously measured during total occlusion of the IRA with an inflated balloon. Changes in echocardiographic wall motion score index (ΔWMSI) and left ventricular end diastolic volume (ΔEDV) were assessed at 4 weeks.
RESULTS: The results of a canonical correlation analysis showed that FCF during AMI was inversely related to ΔWMSI (r=-0.672; p=0.05) and ΔEDV (r=-0.783; p=0.02), but no